

our patient indicated a haemorrhagic tumour which was confirmed at surgery. The histological findings suggest that some of the blood vessel walls in the tumour and connective tissue stroma underwent degenerative changes and ruptured. We suggest that craniopharyngioma be added in the differential diagnosis of parasellar haemorrhagic masses.

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Normal pressure hydrocephalus and cerebrovascular disease: findings of post-mortem

Sir: The clinical distinction between some patients with Alzheimer's disease, multi-infarct dementia and normal (or intermittently raised) pressure hydrocephalus is often difficult. Many demented old people show a combination of changes typical both of Alzheimer's disease and cerebral infarcts at necropsy, hence the term "combined dementia".^{1,2} Similarly, hypertensive cerebrovascular disease and hydrocephalus may co-exist but there is a paucity of confirmatory neuropathological data in the literature.^{3,4} We report the clinical and pathological features in a patient whose dementia initially

resolved following insertion of a shunt for hydrocephalus but who subsequently deteriorated despite a functioning shunt. At necropsy he was found to have multiple small infarcts, particularly in the white matter of the cerebral hemispheres, in addition to periventricular gliosis.

This 66 year old man with mild hypertension presented in 1981 with a 2 year history of clumsiness, falling and memory loss. CT revealed enlargement of all four ventricles with no periventricular lucency. Intraventricular pressure monitoring showed B waves for 43% of the 21 hour period of measurement. Following insertion of a ventriculo-atrial shunt with a low pressure valve in May 1981 the patient improved sufficiently to become independent when walking and managing his financial affairs. CT at this time showed that the ventricles had returned to normal size. He began to deteriorate in 1983 when repeated CT again showed normal sized ventricles. He continued to deteriorate. By 1984 CT showed recurrent ventricular enlargement, now with periventricular lucency. However an isotope shuntogram confirmed that not only was the shunt patent but that there was a normal clearance of isotope through it. The patient subsequently died following a protracted illness that included removal of a small right frontal intracerebral haematoma, and repeated chest and urinary tract infections.

At necropsy (October 1984) the heart was normal in weight (320 g), with no left ventricular enlargement. Moderate atherosclerosis was seen in the coronary arteries with no myocardial infarction. There was thrombus around the distal end of the shunt catheter but the catheter itself was patent. Atherosclerosis was minimal in the thoracic aorta but ulcerated with mural thrombosis in the abdominal aorta. The carotid arteries showed little atherosclerosis and no ulceration. The vertebral arteries were normal. The kidneys contained multiple acute pyelonephritic abscesses and the lungs showed bronchopneumonia. There was a small adrenal cortical adenoma.

The brain weighed 1355 g and showed little cerebral atrophy. A minor degree of atherosclerosis was seen in the basal vessels and the right vertebral artery was hypoplastic. There was no obvious thickening of the meninges and the foramina of Luschka were patent. The lateral ventricles were widely dilated. Histology revealed no senile plaques or neurofibrillary tangles in the hippocampus or frontal cortex. Multiple old and recent infarcts were present in the white matter of the left frontal lobe accompanied by widespread moderately severe arteriosclerosis in

the blood vessels in the same area. There were small numbers of macrophages containing iron pigment in the perivascular spaces. État criblé was present. An old cavity was seen in the right frontal lobe with surrounding gliosis where the haematoma had been removed. Small numbers of old haemorrhages were seen in the right thalamus well posterior to the damaged frontal lobe. A moderate amount of periventricular gliosis was seen which was probably due to the hydrocephalus. There was a mild degree of degeneration of both lateral corticospinal tracts in the spinal cord, more on the right than the left. There was also some mild degeneration of the gracile tracts.

When confronted with a patient with dementia, gait dyspraxia and incontinence, we suggest that it is more realistic to look for a remediable hydrocephalic component to the illness than to consider that patients must have either Alzheimer's disease, multi-infarct dementia or normal pressure hydrocephalus alone. This concept certainly helps when counselling both the patient and his relatives. It is an interesting question whether successful treatment of such hydrocephalus will retard progression of any accompanying damage due to cerebrovascular disease.

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Hypergeusia as the presenting symptom of a posterior fossa lesion

Sir: Hypergeusia (increased sensitivity of taste) is rarely a manifestation of

neurological diseases.¹ We describe a patient with cerebellar tumour who developed bilateral hypergeusia. The possibility of hypergeusia as a symptom of a posterior fossa lesion is emphasised.

A 73 year old man was admitted to our hospital because of taste and gait disturbances. He was well until two months earlier, when he noted that food was too sweet and salty. His wife cooked the food and did not increase doses of sugar and salt for flavour. The patient related that the food tasted two to three times as sweet and salty as before. His wife tried to season the food with smaller doses of sugar and salt. However, his taste disturbance persisted. One month before admission, unsteady gait developed and slowly progressed.

On examination the patient was alert and oriented. The taste disturbance persisted. Mild dysphagia and poor gag reflex were present. Numbness was present in the fingers and toes of the four limbs. There was mild right hemiparesis. Marked truncal ataxia was present with mild ataxia in the four limbs. Deep tendon reflexes were increased in the right limbs, and plantar reflexes were flexor. There were no clinical signs and symptoms of adrenocortical insufficiency. Taste was examined by a filter-paper disc method that measures threshold of taste,² namely, small filter-paper discs (5 mm in diameter) are impregnated with diluted solutions (five gradations) of the four cardinal taste substances: sugar, salt, tartaric acid and quinine hydrochloride and used for qualitative and semi-quantitative tests. He recognised bilaterally the tastes at the lowest concentrations (8×10^{-4} molar for sugar, $5 \cdot 1 \times 10^{-3}$ molar for salt, $1 \cdot 3 \times 10^{-4}$ molar for tartaric acid and $2 \cdot 5 \times 10^{-6}$ molar for quinine hydrochloride). The results indicated lowered threshold of the four qualities compared with Japanese controls.² One week after the first examination, retesting of taste function showed similar results.

Routine laboratory examinations were normal. Plasma cortisol and ACTH values were also normal. A head CT revealed a mass lesion involving the right brachium pontis and right cerebellar hemisphere with a displacement of the brainstem. Biopsy from the right cerebellar hemisphere showed the tumour to be a glioblastoma multiforme. The taste disturbance lasted until radiation and steroid therapy was started.

Hypergeusia is rare and has been given little attention. Rollin¹ reported one patient with multiple sclerosis who had unilateral hypergeusia and hyperpathia on one side of the body. But the responsible lesion was not clear. In adrenocortical insufficiency, the

taste threshold is lowered.³ Aspirin has been reported to increase the perceptibility of bitter taste.³ Clinical data of our case indicated that adrenocortical insufficiency was not likely and aspirin was not given. All taste fibres are distributed to the nucleus of the solitary tract in the medulla oblongata. The central gustatory pathway from the nucleus ascends without decussation in the homolateral pontine tegmentum.⁴ CT showed a displacement of the brainstem by the cerebellar tumour. We could not attribute the hypergeusia to any other cause except for the posterior fossa lesion. Although the mechanism is not known, hypergeusia may be a manifestation of a posterior fossa lesion.

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Palilalia as a symptom of levodopa induced hyperkinesia in Parkinson's disease

Sir: Souques¹ reported on a particular disturbance of language in a patient with stroke leading to left-sided hemiplegia, which presented as compulsive repetition of semantically adequate answers to the examiners' questions. Neither dysarthria nor aphasia affected the speech of the subject. This symptom, termed palilalia by Souques, was also observed in postencephalitic Parkinsonism, but on the whole it must be considered a

rather rare symptom of this disease (for reviews see refs 2, 3). Postmortem examinations have suggested lesions of the striatum as the anatomical substratum of palilalia.⁴

In the cases reported so far palilalia was either constantly present or varied in degree; it occurred both in spontaneous speech and in replying to questions; the number of repetitions usually ranged between four and eight. Reiterations comprised syllables, words, or sentences. Often the verbal repetitions tended to be uttered with increasing rapidity and decreasing loudness.

Besides sporadic manifestations within postencephalitic Parkinsonism, palilalia has also been observed in pseudo-bulbar palsy, Gilles de la Tourette syndrome, Pick's disease, traumatic lesions of the basal ganglia, idiopathic bilateral cerebral calcinosis or in syndromes of unknown aetiology.⁵⁻⁸ As far as we know, the occurrence of palilalia has hitherto not been documented in idiopathic Parkinsonism; rather palilalia was considered a pathognomonic sign of postencephalitic Parkinsonism.¹⁰ In the following report we describe palilalia in a patient with idiopathic Parkinson's disease, manifesting itself in temporal relationship to peak-dose hyperkinesia.

The now 68 years old patient suffered from idiopathic Parkinson's disease of a predominantly akinetic-rigid symptomatology since 1974. In the recent years fluctuations in motor performance, manifesting as frequent shifts from akinesia to choreic hyperkinesia and dystonia developed under treatment with levodopa (800-1200 mg per day, administered usually in eight single doses) and bromocriptine (20-30 mg per day). No tremor was present. Neurological examination revealed no other pathological findings. CT showed minimal cortical atrophy.

Our observations were made in a series of three speech examinations performed at 3-months intervals. Each examination lasted for between 40 and 50 minutes and included the recording of a stretch of spontaneous speech and of word- and sentence-repetitions as well as tests of oral diadochokinesis (rapid syllable repetitions) and sustained phonation. The first and third investigation started after intake of 125 mg levodopa, this dose being part of the daily medication schedule. In contrast, the second examination began about one hour after drug intake. Before drug intake the patient presented with mild akinetic symptoms. His speech was characterised by uniform pitch and loudness, accelerated tempo and sometimes imprecise articulation. Often, the patient did not wait until the examiner had finished with his